Abstract of the Disclosure

The present invention relates to the study and control of atherosclerosis through the modulation of LDL-proteoglycan binding at Site B (amino acids 3359-3369) of the apo-B100 protein in LDL. The invention encompasses methods of identifying compounds which modulate LDL-proteoglycan binding, methods of identifying compounds which modulate atherosclerotic lesion formation, and methods of modulating the formation of atherosclerotic lesions. The invention also encompasses mutant apo-B100 proteins and LDL which exhibit reduced proteoglycan binding while maintaining LDL-receptor binding, polynucleotides which encode these apo-B100 proteins, as well as cells and animals which express the mutant apo-B100 proteins.